Environmental Tobacco Smoke and Periodontal Disease in the United States

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ABSTRACT

Objectives. Cigarette smoking is a leading risk factor for periodontal disease. This cross-sectional study investigated the relation between environmental tobacco smoke (ETS) and periodontal disease in the United States.

Methods. Data were obtained from the Third National Health and Nutrition Examination Survey (1988–1994). The outcome was periodontal disease, defined as 1 or more periodontal sites with attachment loss of 3 mm or greater and a pocket depth of 4 mm or greater at the same site. Exposure to ETS at home and work was self-reported. The study analyzed 6611 persons 18 years and older who had never smoked cigarettes or used other forms of tobacco.

Results. Exposure to ETS at home only, work only, and both was reported by 18.0%, 10.7%, and 3.8% of the study population, respectively. The adjusted odds of having periodontal disease were 1.6 (95% confidence interval = 1.1, 2.2) times greater for persons exposed to ETS than for persons not exposed.

Conclusions. Among persons in the United States who had never used tobacco, those exposed to ETS were more likely to have periodontal disease than were those not exposed to ETS. (*Am J Public Health.* 2001;91:253–257)

Exposure to environmental tobacco smoke (ETS), also known as passive smoking, is the third leading preventable cause of death in the United States—surpassed only by cigarette smoking and alcohol use.[1] ETS contains more than 4000 chemicals, including nicotine and at least 40 known carcinogens.[2] It has been estimated that ETS exposure is responsible for 53 000 deaths annually in the United States.[1,3] Because cigarette smoking is a major risk factor for coronary heart disease and lung cancer, it is not surprising that studies have also linked ETS to these 2 diseases.[1,4–8] However, a causal relation between ETS and coronary heart disease is not yet universally accepted in the scientific community.

In addition to the association between ETS exposure and heart disease and cancer, ETS exposure has been linked to developmental and respiratory effects. In its final report on the health effects of ETS, the California Environmental Protection Agency reported that ETS exposure was causally associated with low birthweight and sudden infant death syndrome.[9] The agency also reported that among children, ETS exposure was causally associated with asthma induction and exacerbation, middle ear infections, chronic respiratory symptoms, and acute lower respiratory tract infections such as bronchitis and pneumonia.[9] Evidence also suggested a causal association between ETS exposure and spontaneous abortion, adverse effects on cognition and behavior, exacerbation of cystic fibrosis, decreased pulmonary function, and cervical cancer.[9] With time, ETS likely will be causally linked to other diseases, especially diseases already linked to cigarette smoking.

One disease that has the potential for such an association is periodontal disease, an infectious disease that destroys the soft tissues and bone supporting the teeth. Cigarette smoking is an important, if not the most important, risk factor for periodontal disease.[10–24] Cigarette smokers are up to 5 times more likely than nonsmokers to develop severe periodontitis.[21] Approximately half of the cases of periodontitis in individuals younger than 30 years are thought to be associated with cigarette smoking.[21] Even though periodontal disease is an infectious disease caused by bacteria, cigarette smoking is believed to increase individuals' susceptibility to periodontal pathogens and tissue destruction. Potential mechanisms for the effect of smoking on periodontal disease include immunosuppression and exaggerated inflammatory cell responses.[25] Recent reports of associations between periodontal disease and systemic diseases such as coronary heart disease,[26–33] stroke,[30] preterm low-birthweight babies,[34,35] and respiratory diseases[36,37] have provided an impetus for identifying new risk factors for periodontal disease and learning more about its pathogenesis.

Only 1 report has been published on the relation between ETS and periodontal disease.[38] The study reported a strong relation between passive smoking in the home and periodontal disease but did not assess workplace exposure or take cigarette smoking into account, even though it included cigarette smokers. The objective of the present study was to examine the association between ETS exposure in the home and at work and the prevalence of periodontal disease among persons who had never used tobacco.

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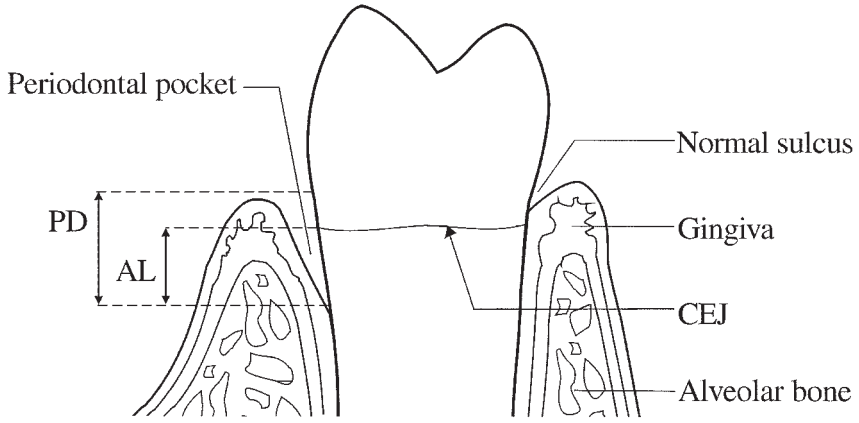
This article was accepted May 17, 2000.

Note. This study was reviewed by the University of North Carolina School of Dentistry Committee on Investigations Involving Human Subjects, approved at minimal risk, and declared exempt from further review.



Note. Pocket depth (PD) is the distance from the gingival margin to the bottom of the sulcus. Attachment loss (AL) is measured from the cementoenamel junction (CEJ) to the bottom of the sulcus.

FIGURE 1—Normal gingival sulcus and a periodontal pocket.

Methods

Study Data and Design

Data for this study were obtained from the Third National Health and Nutrition Examination Survey (NHANES III) conducted from 1988 to 1994. The survey was the seventh in a series of national surveys designed to provide estimates of the health status of the US population. The NHANES III used a complex cross-sectional survey design to sample the total civilian, noninstitutionalized population 2 months or older.[39] A complete description of the plan and operation of the NHANES III may be found elsewhere.[40]

In all, 30818 people were examined, and of these, 15481 persons 13 years or older received periodontal examinations. The present study was limited to the 6611 persons 18 years or older who were assessed for both periodontal disease and ETS exposure and who reported that they had never smoked cigarettes (<100 cigarettes in a lifetime) or used other forms of tobacco (pipes, cigars, and smokeless tobacco). Former and current smokers were excluded because it seemed likely that active smoking would overwhelm any effect of passive smoking.

Measurement of Periodontal Disease

The presence of 1 or more periodontal sites with an attachment loss (i.e., the distance from the cementoenamel junction to the bottom of the sulcus) of 3 mm or greater and a pocket depth (i.e., the distance from the free gingival margin to the bottom of the sulcus) of 4 mm or greater (at the same site) was used as an indicator of periodontal disease (Figure 1). In the NHANES III, 6 dentists trained in the use of National Institute of Dental and Craniofacial Research epidemiologic indices for oral health performed the periodontal examinations.[41] For each subject, a maximum of 7 teeth in randomly selected half-mouths (1 upper and 1 lower quadrant of teeth) were chosen for examination. Examiners used a periodontal probe to measure the distance from the free gingival margin to the cementoenamel junction and the distance from the free gingival margin to the bottom of the sulcus at the mesiofacial and midfacial aspects of each tooth. Third molars, partially erupted teeth, and retained root fragments were excluded from the periodontal examination. Although attachment loss alone is often used in studies as an indicator of periodontal disease, this study required that a tooth site have attachment loss of 3 mm or greater in the presence of a pocket depth of 4 mm or greater to better ensure the presence of active disease. The rationale for this requirement is further explained in the "Discussion" section.

Measurement of ETS Exposure

The independent variable of interest was ETS exposure in the home or at work, dichotomized as yes or no. The NHANES III question "Does anyone who lives here [home of the respondent] smoke cigarettes in the home?" was used to assess home exposure. The NHANES III question "At work, how many hours per day are you close enough to people who smoke so that you can smell the smoke?" was used to assess work exposure. For this study, a subject was classified as having an ETS exposure if 1 or more persons smoked in the subject's home or if the subject had 1 or more hours of smoke exposure at work. Subjects who did not work were included in the analysis and were assessed for home exposure only.

Covariates

Established risk factors for periodontal disease were selected as covariates to be used in the multivariate analysis. The covariates were age at interview, race/ethnicity, sex, education, poverty index, history of diabetes, and dental visits. The poverty index, defined in NHANES III as the family income divided by the poverty threshold for the year in which the family was interviewed,[42] was dichotomized above and below the median value for the study population. History of diabetes was derived from the NHANES III variable "Have you ever been told by a doctor that you have diabetes?" Females who were diabetic only during pregnancy were not considered diabetic. Dental visits were determined from the NHANES III variable "How often do you go to the dentist or hygienist?"

Statistical Analyses

A table showing the distribution of subjects stratified by the study variables and periodontal disease was generated. Crude associations between the study variables and periodontal disease were assessed with χ^2 tests. Unadjusted odds ratios for these associations were estimated with logistic regression.

The adjusted odds ratios and 95% confidence intervals (CIs) for ETS were estimated in a multivariate logistic regression model containing all of the covariates. Covariates were retained in the model regardless of their statistical significance. Two-way interaction terms between ETS and the covariates were tested separately in a fully adjusted model and retained in the model if they reached statistical significance at the .05 level.

All statistical analyses were conducted in SUDAAN, Release 7.50 (Research Triangle Institute, Research Triangle Park, NC). To adjust for the effects of the survey design and to produce unbiased estimates of SEs, the study design variables "total NHANES III pseudo–primary sampling unit" and "total NHANES III pseudo-stratum" were used. Frequencies reported in this article are unweighted; however, all percentages, means, and odds ratios were weighted with the variable "total mobile examination center–examined sample final weight"; thus, the reported values represent values for the target population.

Results

Prevalence of Periodontal Disease and ETS Exposure

The prevalence of periodontal disease in the study population, which consisted of people who had never used tobacco, was 8.8% (SE = 0.65). Among persons who had periodontal disease, an average of 9.7% of the periodontal sites per person had attachment loss of 3 mm or greater and pocket depths of 4 mm or greater.

The overall prevalence of ETS in the home or at work among these nonsmokers was 32.8% (SE = 1.58). Exposure to ETS at home only, work only, and both was reported by 18.0% (SE = 1.10), 10.7% (SE = 0.70), and 3.8% (SE = 0.51) of the population, respectively (0.2% had a missing observation for either work or home exposure, but not both). Within the 2 weeks before the interview, 72% of the study population had worked at a job or business.

Bivariate Associations

ETS exposure and each of the covariates, with the exception of sex, were associated with the prevalence of periodontal disease ($P \leq .05$) (Table 1). The unadjusted odds of having periodontal disease were 1.41 (95% CI = 1.05, 1.90) times greater for persons exposed to ETS than for persons not exposed to ETS. Among this study population, which consisted of persons who had never smoked cigarettes or used other tobacco products, periodontal disease was more likely to be seen in older persons than in younger persons, in Blacks and Mexican Americans than in Whites, in persons with a history of diabetes than in persons without a history of diabetes, and in persons who visited the dentist only when needed than in persons who made visits at least annually. Higher levels of education and income were associated with a lower prevalence of periodontal disease.

Multivariate Model

Adjusted for the covariates, the odds of having periodontal disease were 1.57 (95% CI = 1.15, 2.16) times greater in persons exposed to ETS than in persons not exposed to ETS. In the multivariate model (Table 2), each of the covariates, with the exception of poverty index and history of diabetes, was associated with the prevalence of periodontal disease. None of the 2-way interaction terms between ETS and the covariates were statistically significant and thus were not retained in the model.

TABLE 1—Bivariate Distribution of Persons, by Study Variables and Periodontal Disease[a]

	n[b]	Prevalence of Periodontal Disease, % (SE)	χ^2 *P*	Crude OR (95% CI)
ETS exposure				
No	4371	7.9 (0.74)		1.00 (reference)
Yes	2240	10.8 (1.11)	.029	1.41 (1.05, 1.90)
Age, y				
18–29	2429	4.3 (0.61)		1.00 (reference)
30–49	2380	9.4 (0.81)		2.31 (1.65, 3.22)
50–69	1133	13.2 (1.82)		3.40 (2.36, 4.91)
≥70	669	17.0 (2.79)	.000	4.56 (2.88, 7.22)
Sex				
Female	4422	8.5 (0.78)		1.00 (reference)
Male	2189	9.5 (0.91)	.390	1.12 (0.86, 1.46)
Race/ethnicity				
Non-Hispanic White	2074	6.7 (0.76)		1.00 (reference)
Non-Hispanic Black	1917	17.5 (1.59)		2.96 (2.23, 3.95)
Mexican American	2259	11.6 (0.83)		1.83 (1.38, 2.43)
Other	361	10.2 (1.74)	.000	1.58 (0.97, 2.59)
Education, y				
<12	2259	15.5 (1.58)		1.00 (reference)
12	2156	9.8 (1.21)		0.59 (0.43, 0.82)
>12	2150	5.5 (0.52)	.000	0.32 (0.25, 0.40)
Poverty index[c]				
0.0–1.9	3110	12.4 (0.98)		1.00 (reference)
2.0–11.9	2866	6.9 (0.71)	.000	0.52 (0.41, 0.66)
History of diabetes				
No	6280	8.5 (0.63)		1.00 (reference)
Yes	323	20.6 (3.80)	.001	2.81 (1.81, 4.36)
Dental visits				
At least once per year	2881	6.1 (0.79)		1.00 (reference)
Every 2 years	182	9.6 (2.21)		1.65 (1.06, 2.55)
<Every 2 years	87	9.8 (3.71)		1.68 (0.65, 4.34)
As needed/other	3115	13.0 (0.93)	.000	2.32 (1.71, 3.16)

Note. OR = odds ratio; CI = confidence interval; ETS = environmental tobacco smoke.
[a]Defined as 1 or more periodontal sites with both an attachment loss of 3 mm or greater and a pocket depth of 4 mm or greater.
[b]Unweighted number of subjects. Totals of less than 6611 are the result of missing observations for those variables.
[c]Family income divided by the poverty threshold adjusted for the calendar year in which the family was interviewed.

Discussion

The major finding of this study was that among persons in the United States who had never smoked cigarettes (or used pipes, cigars, or smokeless tobacco), the odds of having periodontal disease were 1.6 times greater for persons exposed to ETS than for persons not exposed to ETS, after control for known risk factors for periodontal disease.

Because both ETS and cigarette smoke are produced by burning tobacco, it would seem plausible that ETS and active cigarette smoking would affect periodontal disease through common mechanisms. Cigarette smoking influences periodontal disease through a variety of local effects (i.e., effects acting directly on the periodontium) and systemic effects. Local effects include vasoconstriction caused by nicotine and decreased oxygen tension, which creates a favorable subgingival environment for colonization by anaerobic bacteria.[25] Although it is possible that the heat from cigarette smoking could have a local effect on the periodontium, no scientific evidence supports such an effect. Systemic effects include impaired chemotaxis, phagocytosis of both oral and peripheral neutrophils, and reduced antibody production.[25] Evidence indicates that nicotine can alter neutrophil phagocytosis and chemotaxis, suppress osteoblast proliferation, and stimulate alkaline phosphatase activity.[25] (For the interested reader, more extensive reviews of the mechanisms by which cigarette smoking affects periodontal disease can be found elsewhere.[25,43]) Because more ETS is probably inhaled through the nose than through the mouth, ETS likely affects periodontal disease through the systemic mechanisms attributed to cigarette smoking rather than through any local effects.

The most important limitation to the study was its cross-sectional design. Because information on periodontal disease and ETS exposure was collected at the same time, and because only current ETS exposure was assessed,

TABLE 2—Multivariate Logistic Model for the Presence of Periodontal Disease[a] (n = 5658)

	β Coefficient	SE	Adjusted OR (95% CI)	Wald F *P*
Intercept	–3.8915	0.3481	…	…
ETS exposure				
No	0.0000	0.0000	1.00 (reference)	
Yes	0.4534	0.1567	1.57 (1.15, 2.16)	.006
Age, y				
18–29	0.0000	0.0000	1.00 (reference)	
30–49	1.2197	0.1851	3.39 (2.33, 4.91)	
50–69	1.5643	0.2090	4.78 (3.14, 7.27)	
≥70	1.9197	0.2793	6.82 (3.89, 11.95)	.000
Sex				
Female	0.0000	0.0000	1.00 (reference)	
Male	0.3430	0.1560	1.41 (1.03, 1.93)	.033
Race/ethnicity				
Non-Hispanic White	0.0000	0.0000	1.00 (reference)	
Non-Hispanic Black	1.1108	0.1898	3.04 (2.07, 4.45)	
Mexican American	0.3463	0.2123	1.41 (0.92, 2.17)	
Other	0.5488	0.3544	1.73 (0.85, 3.53)	.000
Education, y				
<12	0.0000	0.0000	1.00 (reference)	
12	–0.2601	0.2560	0.77 (0.46, 1.29)	
>12	–0.5724	0.2296	0.56 (0.36, 0.89)	.046
Poverty index				
0.0–1.9	0.0000	0.0000	1.00 (reference)	
2.0–11.9	–0.1404	0.1526	0.87 (0.64, 1.18)	.362
History of diabetes				
No	0.0000	0.0000	1.00 (reference)	
Yes	0.3810	0.2484	1.46 (0.89, 2.41)	.132
Dental visits				
At least once per year	0.0000	0.0000	1.00 (reference)	
Every 2 years	0.4761	0.2710	1.61 (0.93, 2.78)	
<Every 2 years	0.7095	0.5639	2.03 (0.65, 6.31)	
As needed/other	0.5923	0.2031	1.81 (1.20, 2.72)	.032

Note. OR = odds ratio; CI = confidence interval; ETS = environmental tobacco smoke.
[a]Defined as 1 or more periodontal sites with both an attachment loss of 3 mm or greater and a pocket depth of 4 mm or greater.

the temporal relation between these 2 factors is not clear. It is certainly possible that the onset of periodontal disease preceded the exposure to ETS in some subjects.

To increase the likelihood that active periodontal disease succeeded or at least coexisted with the exposure, this study defined a periodontal case as one in which the person had at least 1 tooth site with attachment loss and a periodontal pocket. In many studies, attachment loss alone is used as an indicator of periodontal disease. However, attachment loss, which is generally considered irreversible, is a cumulative measure of periodontal destruction throughout one's lifetime. The presence of attachment loss does not necessarily indicate the presence of active disease.[44] Because of periodontal therapy or gingival recession, a person can have "healthy" gingival sulci of normal depth but have attachment loss. However, because periodontal pockets are generally thought to indicate the presence of active disease,[45] the additional requirement that a site with attachment loss also have a periodontal pocket should help ensure that persons had active periodontal disease and, thus, that the ETS exposure at least coincided with the presence of disease.

A second limitation to the study was that information on ETS exposure and the use of cigarettes (and other tobacco products) was self-reported. Some persons exposed to ETS who indicated that they had never smoked cigarettes may actually have been users of tobacco; however, a comparison of self-reported tobacco use and serum cotinine levels generally confirmed self-reported exposures. We used a threshold of 10 ng/mL of serum cotinine as an indicator of current cigarette smoking[46] and found that only 3.4% of the persons exposed to ETS and 0.8% of the persons not exposed to ETS were likely current smokers (or, possibly, users of other tobacco products), even though they had reported that they had never used cigarettes or other tobacco products.

A final limitation to the study was that persons exposed to ETS may have been more likely than nonexposed persons to have some characteristic not controlled for in this analysis that placed them at higher risk for periodontal disease. For example, persons exposed to ETS may have had poorer health behaviors than persons not exposed to ETS. The variability in frequency of dental visits gives some indication that health behaviors may have differed by ETS exposure. In this study, 63% of the persons not exposed to ETS visited the dentist at least once per year compared with 54% of the persons exposed to ETS; however, controlling for these differences in the multivariate analysis had very little effect on the odds ratio for ETS.

Despite these limitations, the study had considerable strengths, including the large number of variables and subjects, which permits other risk factors to be considered in the analysis of passive smoking; verification of self-reported tobacco exposure based on serum cotinine levels; and the NHANES III sampling methodology, which allows results to be generalized to the US population. When considering the public health implications of the current findings, it is important to weigh the strengths and limitations of this study and to acknowledge the current lack of other published evidence on passive smoking and periodontal disease. On the basis of this study alone, it would be premature to declare that passive smoking causes periodontal disease. However, this study and a study by Pirkle et al.[47] clearly found a high prevalence of exposure to ETS in the US population. This underscores the need for additional studies to confirm the relation between ETS exposure and periodontal disease. Specifically, longitudinal studies of human populations and animal-experimental studies are needed; together, they could provide stronger evidence of a causal role of passive smoking.

In the interim, it appears reasonable to use the current findings to reiterate the known oral health hazards of active tobacco consumption, as promulgated in the policies of the major public health and dental organizations in the United States. Those policies currently are targeted primarily toward elimination of active smoking, and of course, it is active smokers who generate the tobacco smoke inhaled by nearly half of the adults in the US population. Regardless of whether additional studies confirm that ETS is a risk factor for periodontal disease, the high prevalence of exposure to ETS and its known effects on mortality and major systemic diseases should provide additional motivation for dentists and dental hygienists to promote tobacco cessation in their practices—an approach that is underutilized[48] but that has the potential to substantially affect public health among the 50% of smokers who make annual dental visits.[49] □

Contributors

S. J. Arbes Jr planned the study, analyzed the data, and wrote the paper. H. Ágústsdóttir and G. D. Slade developed the study question and contributed to the writing of the paper. G. D. Slade supervised the data analysis.

Acknowledgments

Funding for this study was provided through a grant from the National Institute of Dental and Craniofacial Research (T32 DE 0731).

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